La prescrizione dei neurolettici nell’anziano o del perché non se ne viene fuori.

Luca Rozzini

La prescrizione dei neurolettici nell’anziano o del perché non se ne viene fuori.

Luca Rozzini

A cosa servono?

The term behavioural and psychological symptoms of dementia (BPSD; also termed neuropsychiatric symptoms) describes the heterogeneous group of symptoms and signs of disturbed perception, thought content, mood or behaviour that frequently occur in patients with dementia.

Behavioural symptoms

- Aggressività fisica
- Forte vocalizzazione
- Irrequietezza
- Agitazione
- Wandering

Psychological Symptoms

- Ansia
- Depressione
- Allucinazioni
- Deliri

A chi servono?

Abbiamo teoricamente bisogno di un trattamento in grado di bloccare comportamenti che violino i diritti degli altri o che mettano l'individuo in contrasto significativo con norme sociali o figure che rappresentano l'autorità.

Somministro un farmaco per migliorare la condizione di terzi.

The American Psychiatric Association Practice Guideline on the Use of Antipsychotics to Treat Agitation or Psychosis in Patients With Dementia

Guideline Writing Group

American Psychiatric Association (APA) website

American Psychiatric Association (APA) website

- Enormous burden on patients and families or caregivers and often precipitate nursing home admissions due to caregiver distress.
- Management aims to reduce patient suffering, reduce risk of injury, and improve quality of life (QoL), (Mohamed, et al., 2012).

Costs

- The costs of assessment, treatment planning, and discussions with patients, family, or other surrogate decision makers relate to clinician time.
- The CATIE-AD trial (Rosenheck et al. 2007) examined the cost-effectiveness of antipsychotic treatment for outpatients with Alzheimer's disease and psychosis, aggression, or agitation. Although individuals treated with an SGA showed no difference in quality adjusted life years or functional measures as compared with individuals receiving placebo, there were significantly lower costs in the placebo group.
- We are not aware of studies on the cost-effectiveness of antipsychotic treatment for individuals with dementia in inpatient or nursing facilities or for severely agitated or aggressive individuals who require constant supervision.

Cosa trattano?

Therapeutic Advances in Neurological Disorders

Best practice in the management of behavioural and psychological symptoms of dementia

Olivier Pierre Tible, Florian Riese, Egemen Savaskan* and Armin von Gunten*

2017, Vol. 10(8) 297–309

The clinical presentations of BPSD include apathy, depression, anxiety, delusions, hallucinations, sexual or social disinhibition, sleep–wake cycle disturbances, aggression, agitation and other behaviours considered inappropriate.

There are several instruments to systematically assess the presence and severity of BPSD,15 among which the Neuropsychiatric Inventory (NPI)16 and Behavioral Pathology in Alzheimer's Disease Rating Scale (BEHAVE-AD) are recommended. Some BPSD tend to cluster together, usually into four clusters – that is, the affective, psychotic, hyperactive and apathetic clusters

Due paradigmi:
allucinazione ed agitazione

ALLUCINAZIONI, ALLUCINOSI, CHARLES BONNET SYNDROME

Le *allucinazioni* vengono definite nel DSM-5 come esperienze simil-percettive che si verificano senza uno stimolo esterno e che non sono sotto il controllo volontario, viene inoltre specificato che le allucinazioni devono verificarsi nel contesto di un sensorio integro.

Per *allucinosi* si intende quella percezione allucinatoria della quale il soggetto riconosce la natura patologica. Esse si presentano in condizioni: sia fisiologiche come quelle caratterizzanti i contenuti onirici del sonno e la fase I (addormentamento); sia in condizioni patologiche nelle quali esse risultano allucinazioni elementari (uditive o visive).

Charles Bonnet syndrome: La causa più comune di questa grave perdita è la degenerazione della macula o degenerazione maculare, una malattia dove certe cellule fotosensibili della retina funzionano male causando un lento ingrandimento del "punto cieco" al centro della visione.

Review

Visual Hallucinations in PD and Lewy body dementias: Old and new hypotheses

M. Onofrj[a,b,*], J.P. Taylor[c], D. Monaco[a,b], R. Franciotti[a], F. Anzellotti[a,b], L. Bonanni[a,b], V. Onofrj[d] and A. Thomas[a,b]

Behavioural Neurology 27 (2013) 479–493
DOI 10.3233/BEN-129022
IOS Press

Box 2 *An archetypal clinical model of visual hallucinations: Peduncular hallucinosis.*

Peduncular Hallucinosis (PH) appears with lesions involving one of the cerebral peduncles and at least two reports describe hallucinations and hemiparkinsonism contralateral to the lesion [154, 155]. VH are typically complex and vivid, and "although insight is preserved and patients recognize the unreality of the images" [156], the vivid character of the VH is said at times to have a profound emotional effect on the patient [155,157]. In contrast to CBS, tassellopsies and dendropsies are not seen in PH. In PH the content of VH consists of inanimate objects, but mostly complex kinematic scenes are described. For example, in a report by one patient the room was transformed into a train carriage and then into a train with people walking and airplanes flying from the ceiling [71]. As a theoretical model for VH it is hypothesized that VH in this disease arise from brainstem dysfunction and depend upon Rapid Eye Movement Sleep (REM-S) regulating structures.

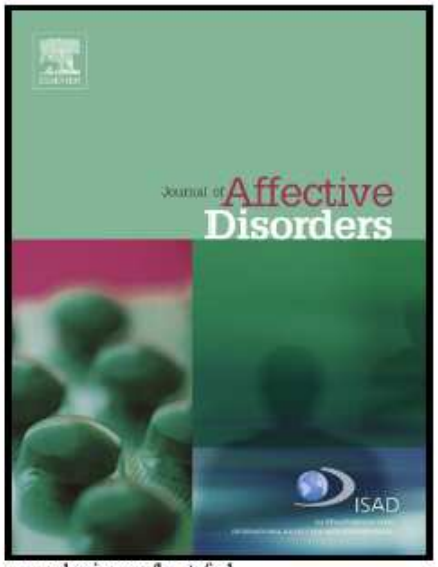
Box 1 *An archetypal clinical model of visual hallucinations: Charles-Bonnet Syndrome.*

Charles Bonnet Syndrome (CBS) patients display VH associated with significant visual loss, with the latter arising as a consequence of cataracts, maculopathy, and optic neuropathy [149–151]. Specific tassellopsies, teicopsies and dendropsies are described in 90% of patients with CBS [152]. More complex hallucinations are also frequent: hallucinations of faces affect 41–47% and hallucinations of people-like figures affect between 40–71% of patients. Hallucinations in this syndrome are traditionally said to be Lilliputian in character [45], although it is now evident that, like in PD, such miniaturization appears only in a minority of CBS patients [54]. A consistently reported feature is preserved insight into the unreality of the hallucinations, but patient interactions with the hallucinations can occur and 41–59% of patient report a strong emotional salience with the occurrence of these hallucinatory phenomena.

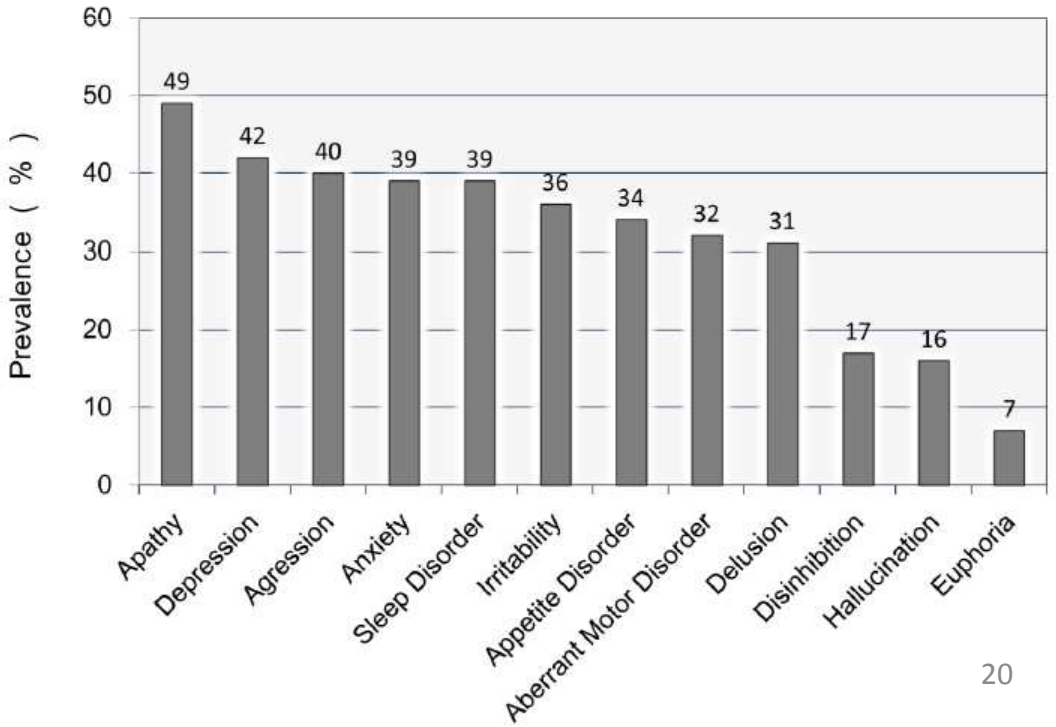
VH in CBS has been simplistically considered to be arising as a result of bottom up deafferentation. However the aetiology of VH in CBS as being simply due to visual loss is currently debated [153] and indeed loss of vision cannot be the sole explanation, as the majority of patients with visual impairment do not experience hallucinations, and hence, other factors are likely to be contributory [153].

The Prevalence of Neuropsychiatric Symptoms in Alzheimer's Disease: Systematic Review and Meta-analysis

Qing-Fei Zhao, Lan Tan, Hui-Fu Wang, Teng Jiang, Meng-Shan Tan, Lin Tan, Wei Xu, Jie-Qiong Li, Jun Wang, Te-Jen Lai, Jin-Tai Yu



Studies published from 1964 to September 30, 2014, were identified from PubMed and Embase database, reference lists and conference abstracts. We calculated prevalence rates and conducted meta-regression analysis with random-effects model, according to study characteristics, population demographics or condition Information. 48 eligible articles.



Pharmacological Management of Lewy Body Dementia: A Systematic Review and Meta-Analysis

Chris Stinton, Ph.D., Ian McKeith, F.Med.Sci., John-Paul Taylor, Ph.D., Louise Lafortune, Ph.D., Eneida Mioshi, Ph.D., Elijah Mak, M.Sc., Victoria Cambridge, Ph.D., James Mason, D.Phil., Alan Thomas, Ph.D., John T. O'Brien, D.M.

AJP in Advance (doi: 10.1176/appi.ajp.2015.14121582)

Antipsychotics

Clozapine. The highest level of evidence for clozapine in PDD was a chart review (PDD, N=8; "other dementia," N=8) (42). Scores on the Brief Agitation Rating Scale and the Cohen-Mansfield Agitation Inventory were significantly lower in the PDD group after treatment (−2.4 and −4.2, respectively), with 62.5% of patients rated as much improved. Side effects included drooling, sedation, tremors, constipation, and delirium. No studies of clozapine for DLB were identified.

Olanzapine. The highest level of evidence for olanzapine was from a secondary analysis of a randomized controlled trial in Alzheimer's disease in which participants were retrospectively identified as meeting DLB criteria and an uncontrolled trial in PDD (43, 44). In participants with possible DLB (N=29), those treated with 5 mg/day of olanzapine (N=10) showed greater reductions in scores on the NPI subscales for delusions (−3.8 points) and hallucinations (−5.9 points) than those receiving placebo (N=10). No significant differences were observed between the placebo group, the 10-mg group, and the 15-mg group (43). While no side effects were reported in that study, other authors have suggested that around 38% of patients with DLB do not tolerate olanzapine even at low dosages (2.5 mg/day) (45). In a sample in which three participants with PDD were treated with olanzapine (44),

Risperidone. The highest level of evidence for risperidone was from a randomized trial in DLB and an uncontrolled trial in Parkinson's disease dementia (50, 51). In participants with PDD and psychosis (N=9) significant reductions were observed in Brief Psychiatric Rating Scale score (−9.5 points) and Cohen-Mansfield Agitation Inventory score (−9.6 points), and improvements were seen in social, occupational, and psychological functioning (Global Assessment of Functioning change, 17 points) (50). No side effects were reported. In DLB, risperidone does not appear to be well tolerated; results of a randomized controlled trial (N=31) suggest deterioration in cognition (MMSE change, −2.3 points), worsening psychiatric symptoms (NPI change, 17.3 points), and study withdrawal (65%) (51).

Quetiapine. The highest level of evidence for quetiapine was from a case series in DLB, a retrospective chart review in PDD, and a randomized controlled trial in Lewy body dementia (47–49). Reductions in psychiatric symptoms were reported for six of nine individuals with DLB following treatment with quetiapine (change in sum of NPI scores on the delusions, hallucinations, and agitation/aggression subscales, 7.7 points) (47). However, 33% of participants withdrew because of adverse events. For individuals with PDD and drug-induced psychosis, quetiapine was associated with worsening cognition and motor function without improvements to psychiatric status (48). A randomized placebo-controlled trial of quetiapine in Lewy body dementia (DLB, N=23; PDD, N=9; Alzheimer's disease with parkinsonian features, N=8) revealed no between-group differences on measures of psychiatric symptoms, cognition, activities of daily living, motor function, or clinician's impression of change (49).

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Relapse Risk after Discontinuation of Risperidone in Alzheimer's Disease

D.P. Devanand, M.D., Jacobo Mintzer, M.D., M.B.A., Susan K. Schultz, M.D., Howard F. Andrews, Ph.D., David L. Sultzer, M.D., Danilo de la Pena, M.D., Sanjay Gupta, M.D., Sylvia Colon, M.D., Corbett Schimming, M.D., Gregory H. Pelton, M.D., and Bruce Levin, Ph.D.

CONCLUSIONS

In patients with Alzheimer's disease who had psychosis or agitation that had responded to risperidone therapy for 4 to 8 months, discontinuation of risperidone was associated with an increased risk of relapse. (Funded by the National Institutes of Health and others; ClinicalTrials.gov number, NCT00417482.)

Agitation in cognitive disorders: International Psychogeriatric Association provisional consensus clinical and research definition

Jeffrey Cummings,[1] Jacobo Mintzer,[2] Henry Brodaty,[3] Mary Sano,[4] Sube Banerjee,[5] D.P. Devanand,[6] Serge Gauthier,[7] Robert Howard,[8] Krista Lanctôt,[9] Constantine G. Lyketsos,[10] Elaine Peskind,[11] Anton P. Porsteinsson,[12] Edgardo Reich,[13] Cristina Sampaio,[14] David Steffens,[15] Marc Wortmann[16] and Kate Zhong[17]

Background: Agitation is common across neuropsychiatric disorders and contributes to disability, institutionalization, and diminished quality of life for patients and their caregivers. There is no consensus definition of agitation and no widespread agreement on what elements should be included in the syndrome.

International Psychogeriatrics (2015), 27:1, 7–17

Agitation is a common clinical manifestation of many neuropsychiatric disorders. It is a frequent manifestation of Alzheimer's disease (AD), frontotemporal dementia (FTD), dementia with Lewy bodies (DLB), and other dementia syndromes (Ballard and Corbett, 2010; Manoochehri and Huey, 2012; Bruns and Josephs, 2013). It occurs in schizophrenia, bipolar illness, and depression (Gonzalez *et al.*, 2013; Swann, 2013). While agitation may include aggressive behaviors, it is not identical to aggression, and agitation can occur without aggression (e.g. pacing, rocking, repetitious mannerisms). Agitation can precipitate institutionalization (Okura *et al.*, 2011), diminishes the quality of life of patients and caregivers (Khoo *et al.*, 2013), and, when severe, may require treatment with medications (Herrmann and Lanctôt, 2007). There is an emerging biology of agitation, and frontal lobe dysfunction is implicated in both clinical and neuroimaging studies (Senanarong *et al.* 2004; Bruen *et al.*, 2008). Treatment of agitation – both pharmacologic and non-pharmacologic – is an unmet need in the care of patients with cognitive impairment (Herrmann and Lanctôt, 2007; Gitlin *et al.*, 2012).

Table 1. Consensus provisional definition of agitation in cognitive disorders

A. The patient meets criteria for a cognitive impairment or dementia syndrome (e.g. AD, FTD, DLB, vascular dementia, other dementias, a pre-dementia cognitive impairment syndrome such as mild cognitive impairment or other cognitive disorder).

B. The patient exhibits at least one of the following behaviors that are associated with observed or inferred evidence of emotional distress (e.g. rapid changes in mood, irritability, outbursts). The behavior has been persistent or frequently recurrent for a minimum of two weeks' and represents a change from the patient's usual behavior.
   (a) Excessive motor activity (examples include: pacing, rocking, gesturing, pointing fingers, restlessness, performing repetitious mannerisms).
   (b) Verbal aggression (e.g. yelling, speaking in an excessively loud voice, using profanity, screaming, shouting).
   (c) Physical aggression (e.g. grabbing, shoving, pushing, resisting, hitting others, kicking objects or people, scratching, biting, throwing objects, hitting self, slamming doors, tearing things, and destroying property).

C. Behaviors are severe enough to produce excess disability, which in the clinician's opinion is beyond that due to the cognitive impairment and including at least one of the following:
   (a) Significant impairment in interpersonal relationships.
   (b) Significant impairment in other aspects of social functioning.
   (c) Significant impairment in ability to perform or participate in daily living activities.

D. While co-morbid conditions may be present, the agitation is not attributable solely to another psychiatric disorder, suboptimal care conditions, medical condition, or the physiological effects of a substance.

NPI ed NPI-4-A/A (Agitazione ed Aggressività)

Ellen B. Dennehy et al, 2012 hanno evidenziato come tra i 12 sintomi comportamentali indagati con NPI l'"agitazione", l'"irritabilità", la "disinibizione" e l'"attività motoria aberrante" consentano di tipizzare il sintomo cluster "agitazione/aggressività" (NPI-4-A/A), che attualmente è di difficile univoca definizione, nei pazienti affetti da Disturbo Neurocognitivo Maggiore secondario a Malattia di Alzheimer (AD).

Benefits and harms of atypical antipsychotics for agitation in adults with dementia

Martin R. Farlow[a,1], Tatyana A. Shamliyan[b,*]

[a]*Department of Neurology, IU Alzheimer's Disease and Related Disorders, 355 W. 16th Street, Suite 4700, Indianapolis, IN 46202, USA*
[b]*Evidence-Based Medicine Quality Assurance, Elsevier, 1600 JFK Blvd 20th floor, Philadelphia, PA 19103, USA*

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We conclude that in difficult-to-manage agitation in adults with progressive dementia, clinicians may recommend atypical antipsychotics with continuous monitoring of behavioral symptoms, informing patients and their families or caregivers of the significant risk of adverse effects and baseline risk of acute myocardial infarction and bone fractures. Future research should shed light on effective and safe treatments for agitation in individual patients with dementia, comorbidities, and baseline risk of mortality and morbidity.

JAMA Clinical Guidelines Synopsis

Antipsychotics to Treat Agitation or Psychosis in Patients With Dementia

Daniel Yohanna, MD; Adam S. Cifu, MD

JAMA September 19, 2017
Volume 318,

MAJOR RECOMMENDATIONS

(1) Outside of situations when patients represent an imminent threat to themselves or others, antipsychotic medications should be used in patients with dementia for the treatment of agitation or psychosis only when symptoms are severe, are dangerous, or cause significant distress to the patient (B recommendation).

(2) The clinical response to non pharmacologic interventions should be reviewed prior to nonemergency use of an antipsychotic medication (C recommendation).

(3) Pharmacologic treatment should be initiated at a low dose and titrated up to the minimum effective dose as tolerated (B recommendation).

(4) If there is no clinically significant response after a 4-week trial of an adequate dose of an antipsychotic drug, the medication should be tapered and withdrawn (B recommendation)

(5) In patients who show adequate response to antipsychotic drug treatment, an attempt to taper and withdraw the drug should be made within 4 months of initiation unless a patient experienced a recurrence of symptoms with prior tapering attempts (C recommendation).

(6) In patients whose antipsychotic medication is being tapered, assessment of symptoms should occur at least monthly during the taper and for at least 4 months after medication discontinuation to identify signs of recurrence and initiate a risk-benefit reassessment of treatment (C recommendation).

(7) In the absence of delirium, if nonemergency antipsychotic medication treatment is indicated, haloperidol should not be used as a first-line agent (B recommendation).

Fanno male?

Antipsychotic Use in the Elderly: Overview and Evidence-Based Management

Helen C. Kales, MD, Mary Blazek, MD, Susan M. Maixner, MD, and Laura M. Struble, PhD, GNP-BC

Vol. 17, No. 1 January 2010 **JCOM**

As compared with research to treat cognitive symptoms, research examining the treatment of NPS of dementia is modest, and no medication is approved by the U.S. Food and Drug Administration (FDA) for this indication.

Nevertheless, conventional antipsychotics have long been used to treat behavioral symptoms, and their overuse in U.S. nursing homes in the 1980s led to federal regulations requiring their oversight.

Introduction of atypical antipsychotics in the 1990s, with lower reported rates of parkinsonism and tardive dyskinesia, there was a significant shift from the use of conventional antipsychotics to atypical.

In 2001, over 70% of atypical antipsychotic prescriptions in the United States were written for off-label indications, and atypical antipsychotics accounted for 82% of antipsychotics written for older patients in Canada in 2002.
At present, atypical antipsychotics have largely replaced conventional antipsychotics as the preferred treatment modality for NPS of dementia.

Tifratene *et al. Alzheimer's Research & Therapy* (2017) 9:34
DOI 10.1186/s13195-017-0256-8

Alzheimer's
Research & Therapy

RESEARCH Open Access

Antipsychotic prescribing for Alzheimer's disease and related disorders in specialized settings from 2010 to 2014 in France: a repeated cross-sectional study

Karim Tifratene[1,2], Valeria Manera[1], Roxane Fabre[1,2], Auriane Gros[1,3], Susanne Thummler[1], Christian Pradier[2], Philippe Robert[1,3] and Renaud David[1,3,4*]

Abstract

Background: Safety warnings from health authorities are currently intended to limit the use of antipsychotics (APs) in dementia-related conditions to treat neuropsychiatric symptoms, such as disturbing and/or delusional behaviors. The aim of this study is to investigate prevalence, correlates and trends of AP prescribing among people with dementia between 2010 and 2014 in the French population.

Methods: AP prescribing and associated factors among individuals with AD, mixed dementia and vascular dementia in the French National Alzheimer Database between 2010 and 2014 were analyzed using multivariate generalized estimating equations models ($n = 199,549$).

Results: In 2014, 7.7% of people with dementia were prescribed an AP. Compared with 2010 there was a 16% increase in AP use. Multivariate analysis showed a linear increase risk of prescription with an adjusted odds ratio (95% confidence interval) of 1.23 (1.17–1.30) in 2014 compared with 2010. Factors associated with AP prescribing were male gender, more severe cognitive decline and living in long-term care facilities. Older age and higher education were protective toward AP prescribing. The type of dementia did not have any influence on AP prescribing.

Conclusion: An increase in AP prescribing among individuals with dementia in French specialized settings over the last 5 years occurred despite safety warnings. This phenomenon suggests that alternative solutions for the management of behavioral and psychiatric symptoms in these populations are still urgently needed.

Keywords: Dementia, Alzheimer's disease, Antipsychotics, Drug prescribing, French Alzheimer Database

does not show that conventional antipsychotics in general or haloperidol in particular increase the risk of mortality in elderly patients

JAMDA 18 (2017) 692–699

JAMDA

journal homepage: www.jamda.com

Original Study

Antipsychotics and the Risk of Cerebrovascular Accident: A Systematic Review and Meta-Analysis of Observational Studies

Wan-Ting Hsu MS [a,b], Amin Esmaily-Fard PharmD [c], Chih-Cheng Lai MD [d], Darshan Zala MS [e], Sie-Huei Lee MD [e,f], Shy-Shin Chang MD, PhD [g,h], Chien-Chang Lee MD, ScD [a,b,*]

A B S T R A C T

Background: Studies investigating the association between antipsychotic use and the risk of cerebrovascular accident (CVA) showed inconsistent results.
Aim: Conduct a systematic review and meta-analysis to evaluate whether use of antipsychotics is associated with increased risk of CVA.
Methods: Major electronic databases were searched from 1970 to October 2016 for observational studies investigating the risk of CVA among users of antipsychotics. Pooled estimates of odds ratios (ORs) and 95% confidence intervals (CIs) were obtained by random effects meta-analysis.
Results: Of 1171 citations identified, 10 studies were considered eligible. Significant increase in risk of CVA was associated with first-generation antipsychotics (OR 1.49; 95% CI 1.24–1.77) but not with second-generation antipsychotics (OR 1.31; 95% CI 0.74–2.30). Use of any antipsychotics in patients with dementia was associated with a low risk of CVA (OR 1.17; 95% CI 1.08–1.26).
Conclusions: The available evidence suggests use of with first-generation antipsychotics as opposed to second-generation antipsychotics significantly increased the risk of CVA.

AP RISK AND DIABETES

The landmark Clinical Antipsychotic Trials of Intervention Effectiveness-Alzheimer's Disease (CATIE-AD) a large double blind RCT study (2006), which detected slight elevations in blood glucose levels among older adult out-patients with dementia who received 3 separate atypical AP compared to placebo over a 36 week period (Schneider, Tariot & Dagerman, 2006).

QT INTERVAL PROLONGATION

QT interval prolongation predisposing patients to arrhythmias and sudden cardiac death is primarily a cumulative effect with other drugs that may prolong the QTc: QTc <450 for men; <460 for women.

There is no clear consensus on the degree of drug-induced QT prolongation that requires drug discontinuation.

(Al-Khatrib, LaPointe, Kramer & Califf, JAMA, 2003).

AP RISK OF FALLS WITH HIP FRACTURES

Meta-analysis of multiple studies of first and second generation antidepressants and AP medications. 166 studies; 10 AP studies, and 14 antidepressant studies with more than 70,000 hip fracture cases. Conclusion: all psychotropic drug classes were associated with an increased risk of hip fractures in older adult populations. Oderda, Young, Asche, & Pepper (2012)

Drug Group	Odds Ratio	95% confidence interval
Conventional AP	1.68	1.43 – 1.99
Atypical AP	1.30	1.14 – 1.49
TCA	1.71	1.43 – 2.04
SSRI, SNRI, bupropion, mirtazapine, trazodone	1.94	1.37 – 2.76

JAMDA

journal homepage: www.jamda.com

Review Article

The Mortality Risk of Conventional Antipsychotics in Elderly Patients: A Systematic Review and Meta-analysis of Randomized Placebo-Controlled Trials

Tessa A. Hulshof RN, MSc [a,b], Sytse U. Zuidema MD, PhD [a], Raymond W.J.G. Ostelo PhD [b,c], Hendrika J. Luijendijk MPH, MD, PhD [a,d,*]

http://dx.doi.org/10.1016/j.jamda.2015.03.015

Results: Data of 17 trials with a total of 2387 participants were available. Thirty-two deaths occurred. The pooled risk difference of 0.1% was not statistically significant (95% confidence interval (CI) −1.0%–1.2%). The risk ratio was 1.07 (95% CI 0.54–2.13). Eleven of 17 trials tested haloperidol (n = 1799). The risk difference was 0.4% (95% CI −0.9%–1.6%), the risk ratio was 1.25 (95% CI 0.59–2.65).
Conclusions: This meta-analysis of placebo-controlled randomized trials does not show that conventional antipsychotics in general or haloperidol in particular increase the risk of mortality in elderly patients. It questions the observational findings and the warning based on these findings.

Teorie sui behavioural disorders in Dementia

Teoria biologica (i.e. corteccia prefrontale mesiale - medial apathetic syndrome, orbito-frontal disinhibited syndrome)

Teoria della personalità (antecedente)

Riduzione della soglia comportamentale: risposte alterate a stimoli

Bisogni non soddisfatti: l'incapacità di gestire esigenze fisiologiche dolore/disconfort fisico/disconfort mentale, solitudine (loneliness), noia

Nonpharmacologic Treatment of Behavioral Disorders in Dementia .
Jiska Cohen-MansfieldFinkel & Burns, 2013

Personality Change in the Preclinical Phase of Alzheimer Disease

Antonio Terracciano; Yang An, MS; Angelina R. Sutin et al.

JAMA Psychiatry. September 20, 2017.

neuroticism, extraversion, openness, agreeableness, and conscientiousness

…individuals who developed dementia scored higher on neuroticism (β=2.83; 95% CI, 1.44 to 4.22; P<.001) and lower on conscientiousness (β=−3.34; 95% CI, −4.93 to −1.75; P<.001) and extraversion (β=−1.74; 95% CI, −3.23 to −0.25; P=.02). ..

Conclusions and Relevance No evidence for preclinical change in personality before the onset of mild cognitive impairment or dementia was identified. These findings provide evidence against the reverse causality hypothesis and strengthen evidence for personality traits as a risk factor for dementia.

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WILEY International Journal of Geriatric Psychiatry

RESEARCH ARTICLE

Self-reported personality traits are prospectively associated with proxy-reported behavioral and psychological symptoms of dementia at the end of life

Angelina R. Sutin[1] | Yannick Stephan[2] | Martina Luchetti[1] | Antonio Terracciano[1]

TABLE 2 Association between self-reported personality traits and proxy-reported behavioral symptoms of dementia

Personality Trait	Behavioral Symptom			
	Lost in Familiar Places	Wander Off	Cannot be Left Alone	Hallucinations
Neuroticism	1.32 (1.10-1.58)**	1.48 (1.11-1.97)**	1.22 (1.01-1.48)*	1.30 (1.09-1.54)**
Extraversion	0.95 (0.78-1.14)	1.23 (0.90-1.67)	0.91 (0.74-1.11)	0.98 (0.82-1.18)
Openness	0.88 (0.72-1.06)	1.01 (0.74-1.37)	0.82 (0.67-1.01)	1.02 (0.85-1.23)
Agreeableness	0.75 (0.61-0.92)**	0.95 (0.68-1.32)	0.90 (0.72-1.12)	1.04 (0.85-1.28)
Conscientiousness	0.74 (0.61-0.90)**	0.92 (0.67-1.25)	0.75 (0.61-0.93)**	0.90 (0.74-1.08)
Sample N	1843	1864	1864	1836
	Depression	Periodic Confusion	Uncontrolled Temper	Symptom Sum
Neuroticism	1.50 (1.29-1.74)**	1.31 (1.12-1.52)**	1.58 (1.32-1.89)**	0.13 (0.09, 0.18)**
Extraversion	0.84 (0.71-0.98)*	1.00 (0.85-1.17)	1.15 (0.95-1.40)	0.00 (−0.05, 0.05)
Openness	1.01 (0.86-1.18)	1.06 (0.90-1.24)	1.15 (0.95-1.40)	0.01 (−0.04, 0.06)
Agreeableness	0.98 (0.82-1.17)	0.95 (0.80-1.14)	0.87 (0.71-1.08)	−0.02 (−0.07, 0.03)
Conscientiousness	0.85 (0.72-1.00)	0.95 (0.80-1.12)	0.83 (0.68-1.01)	−0.06 (−0.11, −0.01)*
Sample N	1941	1984	1990	1740

E’ possibile orientare i comportamenti? Richard Taller attraverso la teoria del nudge (pungolo) o della spinta gentile Quando perdiamo qualcosa l’emozione negativa è molto più forte e persistente di quanto guadagniamo qualcosa.

Therapeutic Advances in Neurological Disorders

Best practice in the management of behavioural and psychological symptoms of dementia

Olivier Pierre Tible, Florian Riese, Egemen Savaskan* and Armin von Gunten*

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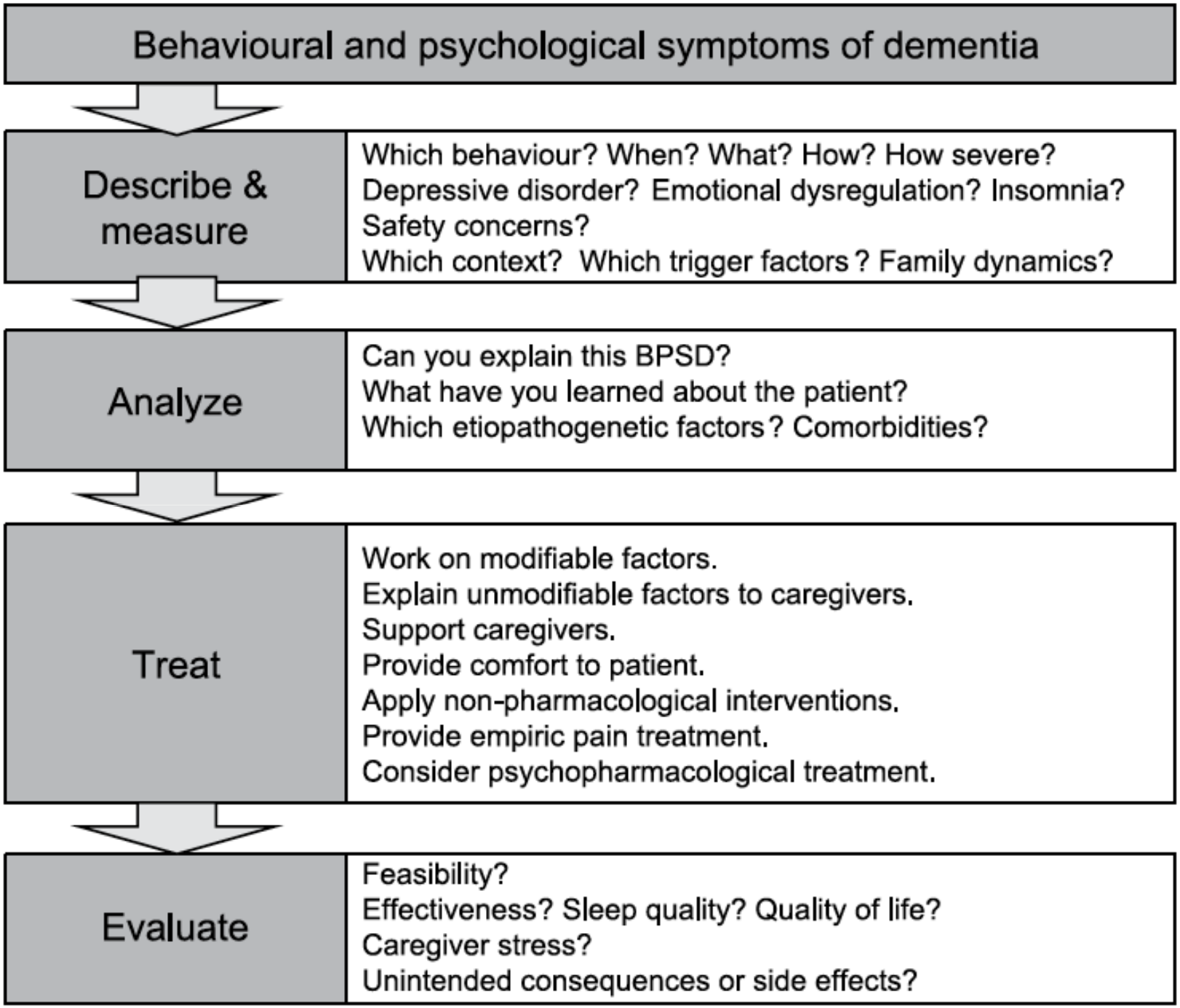
Behavioural and psychological symptoms of dementia
Describe & measure
Which behaviour? When? What? How? How severe?
Depressive disorder? Emotional dysregulation? Insomnia?
Safety concerns?
Which context? Which trigger factors ? Family dynamics?
Analyze
Can you explain this BPSD?
What have you learned about the patient?
Which etiopathogenetic factors ? Comorbidities?
Treat
Work on modifiable factors.
Explain unmodifiable factors to caregivers.
Support caregivers.
Provide comfort to patient.
Apply non-pharmacological interventions.
Provide empiric pain treatment.
Consider psychopharmacological treatment.
Evaluate
Feasibility?
Effectiveness? Sleep quality? Quality of life?
Caregiver stress?
Unintended consequences or side effects?

Può esistere una medicina basata sull'evidenza quando si ha a che fare con manifestazioni eterogenee non scientificamente dimostrabili?
I big data servono?
Per ora il "secondo me" culturale non è l'errore più grosso.